Rotational Atherectomy of a Lesion in Which Stent Expansion Was Limited by Severe Calcification

To the Editor:

Stents implanted into hard, calcified lesions do not always fully expand, even though they are inflated at very high pressure, and once the device has been released the problem is difficult to solve. The ablation of the stent and the calcified plaques protruding through the stent rings by means of high-speed rotational atherectomy reduces wall thickness, facilitating balloon dilation. This technique, which is not without potential risks, has been successfully employed in three patients.1,2

A 70-year-old hypertensive, hypercholesterolemic man was admitted to our hospital for acute coronary syndrome with ST segment depression in anterior leads. The results of physical examination, chest radiography, blood cell count and standard laboratory tests, including serial determinations of creatinine phosphokinase level and troponin T expression, were normal. In addition to the treatment he was receiving at the time of admission, consisting of atenolol, atorvastatin and aspirin, clopidogrel, and intravenous infusion of nitroglycerin was initiated and cardiac catheterization was performed. The left ventricle, which was hypertrophic and presented moderate hypokinesia of the apical region, preserved an ejection fraction of 63%. The bifurcation of the left main coronary artery presented a stenosis of 40% and subtotal occlusion of the ostium of the circumflex artery. This vessel, which was occluded distally, was being supplied via inadequate collateral circulation branching from the right coronary artery, which presented severe stenosis of the middle segment. All these lesions were successfully treated by means of stent placement. The left anterior descending coronary artery (Figure, A), possibly the culprit vessel, presented an extensive severe, calcified stenosis of the middle segment. After predilatation using a 2.5×25-mm Maverick balloon, a 2.75×24-mm Driver stent was implanted; however, a portion of the distal third did not expand completely, presenting a diabolo-like image (Figure, B). An attempt to post dilate using a 3.0×13-mm Powersail balloon inflated to a pressure of 25 atmospheres failed (Figure, C), and therefore rotational atherectomy using a 1.75-mm olive-shaped burr (Figure, D) was carried out. Afterwards, post dilatation resulted effective, and a 3.0×12-mm Taxus pharmacoactive stent was deployed into the previously implanted stent (Figure, E). The final angiographic result was excellent (Figure, F) and there were no adverse events. The postoperative course was satisfactory and the patient, when discharged from the hospital 48 hours later, was asymptomatic. At 5 months, coronary angiography revealed the absence of restenosis in the Taxus stent.

The efficacy of rotational atherectomy in situations of incomplete expansion of stents implanted in severely calcified lesions has been demonstrated in three previously reported cases in which there were no problems associated with the erosion of the metal.1,2 The success may be attributed to the ablation of the stent rings and the calcium that protruded through them, resulting in the thinning of the wall. Although
certain potential complications, such as the distal emboliza-
tion of metal particles or late thrombosis, could develop,
they did not occur in any of the four cases documented in
the literature. Our case confirms the feasibility of this tech-
nique in special cases.

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Unusual Electrocardiographic
Presentation of Arrhythmogenic
Right Ventricular Cardiomyopathy

To the Editor:

Arrhythmogenic right ventricular cardiomyopathy or
dysplasia (ARVD) is a genetically determined heart mus-
cle disease in which the myocardium is replaced by fibroa-
dipose tissue. It is associated with arrhythmias, heart failu-
re and sudden death.1 Typical symptoms include
palpitations, dizziness and syncope. Sudden death is often
the first sign, especially in young people, and it coincides
with some type of strenuous physical activity; in some se-
ries, it accounts for up to 20% of all sudden deaths in
young adults.2 With the introduction of automatic implan-
table defibrillators, many of these deaths can be prevented.
However, the prevention of arrhythmic death requires ti-

mely diagnosis, which depends on the ability of the pri-
mary care physician and the specialist to be aware of and
to diagnose this disease.

The diagnosis is established on the basis of certain
major and minor criteria involving the evaluation of the fa-
mily history, the depolarization or repolarization-induced
electrocardiographic changes, arrhythmias, structural chan-
ges and ventricular dysfunction, and on the his-
topathological features.3 At the present time, there are a
number of registries underway for the purpose of analy-
zing the validity of these criteria and broadening them, if
appropriate.4 The typical clinical signs, together with
T-wave inversion in leads V1 to V3 present in over 50% of
the patients,5 or the appearance of the epsilon wave in
30%,6 raise the suspicion of this disease; however, there
are reports of cases in which the electrocardiographic fea-
tures were less common, but the risk of sudden death was
the same.7 We present the case of a 31-year-old man, a
professional soccer player, who was referred to our unit
because of occasional palpitations. After initial evaluation
and the verification of the absence of a family history of
heart disease, we examined the electrocardiogram, which
revealed negative T waves in leads II, III, and aVF and
ventricular premature beats with right bundle branch block
(RBBB) morphology (Figure 1). During 24-hour Holter
monitoring, several episodes of nonsustained ventricular
tachycardia (NSVT) were recorded, as were premature be-
ats with RBBB morphology that appeared to originate in
the left ventricular apex. Transthoracic echocardiography
revealed a slightly dilated left ventricle with apical akine-
sia. Given these findings, the patient underwent single
photon emission computed tomography (SPECT) to study
myocardial work, reaching 20 MET without symptoms,
but with frequent premature beats and self-limited episo-
des of NSVT similar to those recorded during
24-hour Holter monitoring. The scintigraphic images dis-
closed a fixed apical perfusion defect. In view of these fin-
dings, coronary arteriography was carried out, but no le-
sion of any type was observed; however, ventriculography
revealed the presence of left ventricular apical akinesia.
Right ventricle was dilated and unstructured, with akine-
sic/dyskinetic areas and diastolic septal bulge (“stack of
coins”; Figure 2). As ARVD was suspected, magnetic re-
sonance imaging was requested. It revealed fatty infiltra-
tion of the right ventricle. ARVD with involvement of the
left ventricle was diagnosed.